The International Programme for Chemical Safety has reiterated this position (IPSC, 1998). These conclusions continue to support what the industry said in 1965 that the only safe level to prevent disease is zero and it also supports the finding that non-malignant respiratory diseases need not be present before cancer of the lung or mesothelioma can develop.

There is marked enhancement of the risk of lung cancer in workers exposed to asbestos who also smoke cigarettes (Selikoff et al., 1968; Berry et al., 1972; Hammond and Selikoff, 1973; Hammond et al., 1979). Data from Hammond et al. (1979) and Weiss (1971) suggest cigarette smoking may also contribute to the risk of asbestosis. Smoking, however, has not been found to be associated with an increased risk of pleural or peritoneal mesothelioma, or cancers of the stomach, colon and rectum, which occur with equal frequency among smoking and non-smoking asbestos workers. OSHA attributes asbestos exposure with 79.4 percent of the lung cancer deaths among asbestos-exposed workers who smoke and 77.2 percent of lung cancer deaths among non-smokers (OSHA, 1986).

Toxicity of Short Asbestos Fibers

Any assumption that short fibers, less than 5 micron in length, are not hazardous can not be justified based on the available science. Because the analytical method of choice, for regulatory purposes, has been the phase contrast method [PCM] which counts only fibers greater than 5 um in length epidemiology studies that have been forced to compared doses, in their cohorts, to fibers greater than 5 μm in length. It must be noted that the PCM analytical method was chosen based on its ability to count fibers only and not on a health effect basis. While PCM has been the international regulatory method for analysis it is not able to detect small diameter fibers [<0.2μm in diameter] and because of this, it is suggest that transmission electron microscopy [TEM] should be an adjunct to PCM, since the evidence suggests that PCM may underestimate exposures and the health risks as found in the analysis of brake residue (Yeung, P. et, al., 1999).

To assume that shorter fibers do not cause disease can not be based on the majority of epidemiological studies to date. Stanton & Wrench & Stanton et al. found that the longer, thinner fibers were more carcinogenic, but could not identify a precise fiber length that did not demonstrate biological activity. (Stanton, M.F. & Wrench, C. 1972) & (Stanton, M.F.et al 1981). It must be kept in mind that Dr. Stanton has never said long fibers are bad and short fibers are good, in fact he appreciated that a large number of short fibers, individually of low

tumorogenic probability might be more hazardous than fewer long fibers, individually of high probability (Greenberg, M, 1984). It has been shown that it is not the just the size and shape of the various asbestos fibers that are important in the fiber's ability to produce disease but other factors may play a role in the carcinogenicity of the mineral fiber (Wagner, J.C.(ed.) 1980) & Wylie, A.G.et al. 1987). Studies have also found that the majority of asbestos fibers in lung and mesothelial tissues were shorter than 5 μm in length, thus indicating the ability of the shorter fibers to reach the tumor site, remain there, and therefore their role in the etiology of disease is implicated (Suzuki,Y. & Yuen, S.R. 2002) & (Dodson, et al. 2001). NIOSH research has found that in typical occupational environments fibers shorter than 5 μm in length outnumber the longer fibers by a factor of 10 or more (Dement, JM & Wallingford, KM, 1990). Shorter fibers must be studied in more depth and short fibers should not be disregarded especially when clearance is retarded (Oberdorster, G. 2001). That chrysotile fibers tend to spit longitudinally as well as partially dissolve, resulting in shorter fibers within the lung, was reported in a review of several articles (Dement, JM & Brown, DP, 1993). Additionally, Fubini argues that, because all asbestos appear nearly equally potent, length and fiber form do not appear influential on the outcome of disease (Fubini, B. 2001). Fubini makes this conclusion based on work of Boffeta et al., which concludes that the specific type of asbestos is not correlated with lung cancer risk but that industry specific exposure appears to fit the linear slope best, a finding also supported by Dement & Brown (Boffetta, P. 1998). For mesothelioma, induction was related to the time since first exposure and potency with both industry type and asbestos type (Boffetta, P. 1998).

The Agency for Toxic Substances and Disease Registry (ATSDR), in response to concerns of short asbestos fibers resulting from the collapse of the World Trade Towers, asked a contractor to convene a panel of seven experts to evaluate the role of short fibers with human disease potential (ATSDR, 2003). As to non-carcinogenic lung diseases associated with short asbestos fibers the report concludes that "...short fibers may be pathogenic for pulmonary fibrosis, and further research is needed to clarify this issue." The panel concluded that for carcinogenic effects of short fibers the current weight of the evidence is that short fibers less than 5 micron "...are unlikely to cause cancer in humans." While these conclusions were found in the executive summary of the report, a more in-depth review of the body of the report points to a less conclusive assessment for the role of short fibers in the etiology of cancer. In fact, in panel discussions it was noted that no epidemiologic studies have examined populations exposed only to short asbestos fibers. One epidemiology study that may have the ability to address this issue suffered from short latency to evaluate the development of cancer (Higgins,ITT,et al.,1983). Another study of workers having exposure for at

least 5 years, in a gold mine with 94% of the asbestos fibers being less than 5 μm in length, found an increased mortality from respiratory cancers [10 obs. vs. 2.7 exp.: SMR 3.7; 95% CI = 1.78 - 6.81] and non-malignant respiratory diseases [8 obs. vs. 3.2 exp.: SMR 2.5; 95% CI = 1.08 - 4.93] (Gillam, et al., 1976). A subsequent study (McDonald et al., 1978). looking at the same mine reported above, of miners with 21 or more years underground experience, did not find such an increase for respiratory cancers but did for non-malignant respiratory disease however, when analyzing the data from the previous study for only those with 20 years or greater years of employment, both respiratory cancers [7 obs. vs. 2.18 exp.: SMR 3.2; 95% CI = 1.29 - 6.62] and non-malignant respiratory diseases [8 obs. vs 2.56; SMR 3.1; 95% CI = 1.35 - 6.16] were still significantly increased. Two other studies of miners, where 38% of the asbestos fibers were shorter than 5μm in length also found excess mortality from lung cancer, mesothelioma as well as non-malignant respiratory disease and that the mortality patterns for mesothelioma were significant because they were much greater than that of crocidolite miners in South Africa and Australia (McDonald,J.C. et al., 1986).& (McDonald et al. 2002)

Animal studies can be misleading when looking at short fibers, especially as rodents clear short fibers from their lungs at a rate approximately 10 times faster than do humans (ASTDR, 2003). Experimental models are limited also, due to the fact that only fibers of very limited length distributions have been tested (Dodson, RF., et.al., 2003). Further, when appropriate analytical techniques have been used the overwhelming majority of the asbestos fibers in the tissues have been found to be less than 5μm in length (Dodson, RF., et al., 2003). Only two of the seven ATSDR panelists felt there was a reasonable certainty of no harm from short fibers while the other four remained concerned about the ability of short fibers to cause harm (ATSDR, 2003). In fact, tremolite asbestos fibers were found to produce the highest average fibrosis grades when exposures were to average tremolite fibers less than 5μm in length (Nayebzadeh,et. al., 2001).

Building Workers and Occupants

Review of the death certificates from a mesothelioma surveillance system in Wisconsin, since 1959 and a cancer reporting system since 1978, found 487 mesothelioma deaths in Wisconsin from 1959 to 1989 which led to the investigation of 41 persons with likely exposure to in place asbestos-containing building materials (ACBM). The investigation found in the 41 mesothelioma; 12 school teachers, 10 school maintenance employees, 7 public building maintenance workers, 5 private building maintenance workers, and 7 commercial and factory workers performing maintenance activities. After adjusting for all asbestos exposures the

authors concluded that individuals occupationally exposed to in-place ACBM are at risk for the subsequent development of mesothelioma [Anderson et al, 1991). Lilienfeld also reported four cases of malignant mesothelioma in school teachers whose only apparent exposure to asbestos was in the schools in which they taught [Lilienfeld, 1991]. Oliver et al (1991) has also reported a study of 120 public school custodians where they found pleural plaques, an indicator of exposure to asbestos, in 33% of the total group and 21% in those employees with no other known exposures to asbestos but through their school work. They also found more restrictive lung disease and both plaques and restriction occurred with significant associations with duration of employment [Oliver et al, 1991). More recently a French study found similar results to Oliver et al, 1991 related to latency with duration of exposures to building custodial and maintenance employees [Matrat et al, 2004).

Anderson et al. x-rayed 457 school maintenance and custodial workers and found conditions consistent with asbestos-induced diseases including pleural abnormalities which could not be explained to prior work before that of their present occupation. Laborers, at the schools, with more than 20 years of school employment had the highest prevalence of abnormalities [Anderson et al., 1992). Churg & Warnock found asbestos bodies in the lungs of 21 patients in the general population, who had 300 to 9,000 bodies/g, which the authors claim, is a concentration frequently found in manual laborers among the general population who were not primary asbestos workers and conclude that among laborers their risk was most likely occupational in nature, thus confirming that laborers are at risk of asbestos-related disease (Churg & Warnock, 1979). Among 660 custodians, employed by the New York City Board of Education, were examined between 1985 through 1987 for asbestos-related disease and 39% of those with 35 years of employment had abnormal films. 84% reported removing asbestos and 89% reported working in area where asbestos was present and abated [Levin &, Selikoff, 1991). In a study of male employees of one California school district 13.3% of custodian were found to have asbestos-related disease and because these were related to parenchymal and pleural fibrosis it would indicated rather high exposures to these custodial workers (Balmes et al., 1991). Among the statistics from the Australian Mesothelioma between 1980-1985 laborers represented the greatest percentage of jobs with mesothelioma (14.8%) [Yeung & Rogers, 2001). Among 11,685 members of the Laborers' International Union of North America (LIUNA), who died between 1985-1988 found statistically significant elevated mortality risks for lung cancer (N = 1208, PCMR1 = 1.06, 95% CI: 1.00-1.12) and 20 mesothelioma deaths (Stern et al., 1995).

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1 PCMR = Proportional Cancer Mortality Ratio.

EPA does not believe that air sampling within buildings can represent the entire picture of potential risk to inhabitants or building workers because they believe it is of limited significance. Because of this EPA does not rely solely on air sampling to determine the necessity for remediation of buildings having asbestos-containing materials [EPA, 1985 & 1990].

Conclusions and Opinions

In order to augment this report I have attached a chronological profile titled "Asbestos Timetables" which I prepare in order to place the information I have presented in this report in a perspective that can easily be viewed over time. This timeline also included additional information concerning the early history of asbestos usage, specific occupational exposures and products from which exposures have occurred. This list is by no means all inclusive, but does give a good overview of the development of knowledge concerning asbestos disease. In addition, a specific analysis of occupational exposure guidelines, TLV's® and government standards are outlined in the timetables.

Specifically, my opinions are as follows:

Asbestos is a harmful substance which can cause both disease and death;

The knowledge of asbestos' harm to humans has been known for decades;

All forms of asbestos can cause all asbestos-related diseases;

All exposures to respirable asbestos fibers contribute to increasing the risk of developing asbestos-related disease;

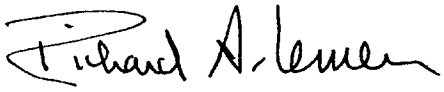
Prevention methods for reducing the risk of disease among asbestos exposed persons have been known since the 1930's;

A safe exposure concentration has never been identified for exposure to asbestos for which cancer will be prevented.

Asbestos-containing materials in occupied buildings can cause a variety of diseases when asbestos particles/fibers are released into the air and inhaled by building occupants including custodial workers, labors, maintenance workers, and teachers.

Asbestos-containing materials can release asbestos particles/fibers can be released into the air when disturbed, during deterioration and during removal.

All of my conclusions are based on my education, training, experience, and review of the medical and scientific literature. I have also included a listing of my trial and deposition testimony as I may have kept them for the last four years (I can't verify their inclusiveness). My fees for trial and deposition testimony are $400.00 per hour.



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